HPS Trailer Page
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EAST

UserID: RShukla_Job_1_of_1

Printer: cm1_12e14_gbleptr

Summary

Document	Pages	Printed	Missed
US20010012514	19	19	0
Total (1)	19	19	0

L Number	Hits	Search Text	DB	Time stamp
1	1842	beta-interferon or beta ADJ interferon	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:38
7	857	muliple ADJ sclerosis or experimental ADJ allergic ADJ encephalomyelitis	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:25
13	4	(beta-interferon or beta ADJ interferon) same (muliple ADJ sclerosis or experimental ADJ allergic ADJ encephalomyelitis)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:25
25	18700	gene ADJ therapy	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:31
31	0	((beta-interferon or beta ADJ interferon).clm.) same (gene ADJ therapy)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:31
37	30	((beta-interferon or beta ADJ interferon).clm.) and (gene ADJ therapy)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:31
43	0	(beta-interferon or beta ADJ interferon) SAME (gene ADJ therpay).clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:39
19	166	(beta-interferon or beta ADJ interferon).clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:43
49	9806	braun$.in	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:44
55	1	((beta-interferon or beta ADJ interferon).clm.) and braun$.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/08/05 15:44

(FILE 'HOME' ENTERED AT 15:07:40 ON 05 AUG 2002)

FILE 'MEDLINE' ENTERED AT 15:07:49 ON 05 AUG 2002

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L1          4083 S INTERFERON BETA OR BETA INTERFERON
L2          4083 S INTERFERON-BETA OR BETA-INTERFERON
L3             6 S BETA-INTERFERON-1A
L4         75180 S MS OR MULTIPLE SCLEROSIS
L5           630 S L2 (S) L4
L6           184 S L2 (S) L4 (S) THERAPY
L7           102 S L6 NOT PY>1999
L8           556 S (BRAUN S?)/AU
L9             0 S L6 AND L8
L10          184 S L6 AND L2
L11          184 S L6 AND L2 AND L4
L12            0 S L8 AND L2 AND L4
L13            0 S L8 AND L2
L14            1 S L6 AND GENE THERAPY
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FILE 'MEDLINE, USPATFULL, PCTFULL, CAPLUS' ENTERED AT 15:15:52 ON 05 AUG 2002

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L15           16 S L14
L16           16 DUP REM L15 (0 DUPLICATES REMOVED)
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L4 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:911121 CAPLUS
DOCUMENT NUMBER: 134:55499
TITLE: Compositions and methods for the treatment or prevention of autoimmune disorders using **DNA vaccine** encoding a self-antigen
INVENTOR(S): Von Herrath, Matthias G.
PATENT ASSIGNEE(S): The Scripps Research Institute, USA
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000078360	A1	20001228	WO 2000-US16218	20000613

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-336672 A 19990617
AB The present invention provides compns. and methods for the prevention or treatment of autoimmune disorders using **DNA vaccine** encoding a self-antigen. In particular, the invention methods utilize plasmid vector encoding at least a portion of an autoreactive epitope that, upon administration to a subject, acts to modulate the immune system thereby ameliorating conditions assocd. with an autoreactive antigen. The compns. and methods of the invention include co-administration of another vector encoding a biol. response modifier (e.g., a cytokine, chemokine, interferon, interleukin) for the effective induction of regulatory cytokines to down-regulate the immune system of a mammal having an autoimmune condition. The invention is exemplified by the treatment or prevention of insulin dependent diabetes in a murine model using RIP-LCMV-NP: transgenic mouse line that expresses lymphocytic choriomeningitis virus nucleoprotein under control of the rat insulin promoter. The exemplary autoreactive epitope used is from insulin .beta. chain. RIP-NP transgenic mice are treated with pCMV-NP with pCMV-ins-B and LCMV-specific CTL responses are evaluated. The studies compare the progression of diabetes in immunized and non-immunized mice and show that the transfer of splenocytes from insulin-B protected mice prevents IDDM and the self-reactive (LCMV-NP) CTL activity in pCMV-B protected mice is reduced.
REFERENCE COUNT: 3
REFERENCE(S): (1) Nicolette, C; WO 9820467 A 1998 CAPLUS
(2) Univ Southern California; WO 9745144 A 1997 CAPLUS
(3) Von Herrath, M; JOURNAL OF IMMUNOLOGY 1998, V161(4), P5087 CAPLUS

L4 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2000:395802 BIOSIS
DOCUMENT NUMBER: PREV200000395802
TITLE: A plasmid construct encoding murine **interferon beta** antagonizes the replication of herpes simplex virus type 1 in vitro and in vivo.
AUTHOR(S): Cui, Bo; Carr, Daniel J. J. (1)

CORPORATE SOURCE: 1. Department of Ophthalmology, Dean McGee Eye Institute, University of Oklahoma Health Sciences Center, 608 Stanton L. Young Boulevard, Rm 415, Oklahoma City, OK, 73104 USA
SOURCE: Journal of Neuroimmunology, August 1, 2003, Vol. 138, No. 1-2, pp. 98-102. print.
ISSN: 0165-5728.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB In the present study, we employed a plasmid DNA encoding murine interferon (IFN)-beta to assess its antiviral efficacy in an in vitro transfection-infection assay and in an ocular HSV-1 infection model of mice. In the in vitro assay, transfection of mouse fibroblasts with the IFN-beta transgene resulted in a 17-fold or greater reduction in the viral load of HSV-1 at a multiplicity of infection (MOI) of 1 compared to that of those mice treated with the plasmid control. RT-PCR analysis of representative immediate early (ICP27), early (thymidine kinase, TK) and late (VP16) viral genes found no changes in the level of expression comparing the IFN-beta transgene- to the vector-treated control group, suggesting that the IFN-beta transgene may act at the post-transcriptional level of viral replication. In the ocular HSV-1 infection model, topical application of the plasmid DNA encoding murine IFN-beta onto mouse cornea enhanced cumulative survival and significantly reduced the viral load of HSV-1 in the eyes and trigeminal ganglia of mice at both day 3 and 6 post-infection compared with mice treated with the plasmid vector control or normal saline. Neutralizing antibody to IFN-beta blocked the protective effect elicited by the IFN-beta transgene. Unlike the in vitro experiment, viral gene expression was reduced in the trigeminal ganglion of mice pre-treated 24 h with the IFN-beta transgene day 3 (ICP27 and VP16) and day 6 (ICP27, TK, DNA polymerase, and VP16) post-infection in comparison to mice treated with the plasmid vector control as determined by semi-quantitative RT-PCR.

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L6 ANSWER 8 OF 38 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2000:492133 BIOSIS
DOCUMENT NUMBER: PREV200000492254
TITLE: **Gene therapy** for autoimmune disorders.
AUTHOR(S): Evans, C. H. (1); Ghivizzani, S. C.; Oligino, T. J.; Robbins, P. D.
CORPORATE SOURCE: (1) Center for Molecular Orthopaedics, Harvard Medical School, 221 Longwood Avenue, BL-152, Boston, MA, 02115 USA
SOURCE: Journal of Clinical Immunology, September, 2000 Vol. 20, No. 5, pp. 334-346. print.
ISSN: 0271-9142.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Although many autoimmune disorders do not have a strong genetic basis, their treatment may nevertheless be improved by **gene therapies**. Most strategies seek to transfer genes encoding immunomodulatory products that will alter host immune responses in a beneficial manner. Used in this fashion, genes serve as biological delivery vehicles for the products they encode. By this means **gene therapy** overcomes obstacles to the targeted delivery of proteins and RNA, and improves their efficacy while providing a longer duration of effect, and, potentially, greater safety. Additional genetic strategies include DNA vaccination and the ablation of selected tissues and cell populations. There is considerable evidence from animal studies that **gene therapies** work: examples include the treatment of experimental models of rheumatoid arthritis, **multiple sclerosis**, diabetes, and lupus. Pre-clinical success in treating animal models of rheumatoid arthritis has led to the first clinical trial of **gene therapy** for an autoimmune disease. In this Phase I study, a cDNA encoding the interleukin-1 receptor antagonist was transferred to the knuckle joints of patients with advanced rheumatoid arthritis. Two additional clinical trials are in progress. It is likely that **gene therapy** will provide effective new treatments for a wide range of autoimmune disorders.

L6 ANSWER 9 OF 38 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2000:492132 BIOSIS
DOCUMENT NUMBER: PREV200000492253
TITLE: **Gene therapy** in experimental autoimmune encephalomyelitis.
AUTHOR(S): Mathisen, Peter M. (1); Tuohy, Vincent K.
CORPORATE SOURCE: (1) Department of Immunology, NB30, Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, OH, 44195 USA
SOURCE: Journal of Clinical Immunology, (September, 2000) Vol. 20, No. 5, pp. 327-333. print.
ISSN: 0271-9142.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Gene therapy** traditionally has been associated with "gene replacement," where exogenous recombinant DNA is introduced ex vivo into somatic cells that are then introduced back into the patient as a way to correct an inherited genetic defect. However, several novel **gene therapy** strategies for treating autoimmune diseases recently have emerged. Strategies involving the use of several types of DNA vaccines, the application of various viral vectors, and the use of diverse cellular vectors have shown promise in inhibiting autoimmune-mediated inflammation and repairing tissue damaged as a result of autoimmune attack. In the current review, we examine and discuss the development and proposed use of emerging **gene therapy** strategies for the treatment of autoimmune disease with specific emphasis

on experimental autoimmune encephalomyelitis (EAE), an animal model widely used in **multiple sclerosis** (MS) research.

L6 ANSWER 11 OF 38 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2000:492087 BIOSIS
DOCUMENT NUMBER: PREV200000492087
TITLE: DNA vaccination in the treatment of autoimmune disease.
AUTHOR(S): Garren, Hideki (1); Steinman, Lawrence
CORPORATE SOURCE: (1) Department of Neurology and Neurological Sciences, Stanford University, Stanford, CA, 94305-5316 USA
SOURCE: Fathman, C. Garrison. Current Directions in Autoimmunity, (2000) Vol. 2, pp. 203-216. Current Directions in Autoimmunity; Biologic and gene therapy of autoimmune disease. print.
Publisher: S. Karger AG CH-4009, Basel, Switzerland.
ISSN: 1422-2132. ISBN: 3-8055-6949-1 (cloth).
DOCUMENT TYPE: Book
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 11 OF 38 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2000:340581 BIOSIS
DOCUMENT NUMBER: PREV200000340581
TITLE: Cytokine **gene therapy** of autoimmune demyelination revisited using herpes simplex virus type-1-derived vectors.
AUTHOR(S): Martino, G. (1); Poliani, P. L.; Marconi, P. C.; Comi, G.; Furlan, R.
CORPORATE SOURCE: (1) Neuroimmunology Unit, San Raffaele Scientific Institute-DIBIT, Via Olgettina 58, 20132, Milano Italy
SOURCE: Gene Therapy, (July, 2000) Vol. 7, No. 13, pp. 1087-1093. print.
ISSN: 0969-7128.
DOCUMENT TYPE: General Review
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The peripheral delivery of drugs in patients affected by central nervous system (CNS)-confined diseases is therapeutically ineffective due to the presence of the blood-brain barrier which forms an inaccessible wall to the majority of CNS targeting molecules. When molecules with an anti-inflammatory profile have been systemically administered to patients affected by a chronic inflammatory demyelinating disease of the CNS, such as **multiple sclerosis** (MS), results have been disappointing. A successful therapeutic approach in MS should therefore consider the delivery of anti-inflammatory molecules directly into the CNS in order to inhibit blood-borne CNS-confined mononuclear cells which act as ultimate effector cells directly destroying oligodendrocytes and/or releasing myelinotoxic substances. Biological and physical vectors engineered with heterologous genes coding for immunomodulatory cytokines with an anti-inflammatory profile might represent the appropriate tool to deliver therapeutic genes into the CNS of patients with MS. So far, cytokine **gene therapy** has never been attempted in MS, but encouraging results have been obtained in the animal model of MS, experimental autoimmune encephalomyelitis (EAE), using viral vectors or plasmids engineered with cytokine genes and then injected systemically, either in the blood stream or circulating encephalitogenic T cells, or into the CNS. Here, we critically discuss the various attempts made in EAE using **gene therapy** protocols based on the delivery of immunomodulatory cytokine genes. Special emphasis is put on the use of non-replicative herpes simplex type-1 (HSV)-derived vectors engineered with the gene of the immunomodulatory cytokine interleukin (IL)-4.